PB86-169893

Scaling the Physiological Effects of Exposure to Radiofrequency Electromagnetic Radiation Consequences of Body Size

(U.S.) Health Effects Research Lab.
Research Triangle Park, NC

Prepared for

Idaho Univ., Moscow

1984

U.S. Department of Commerce
National Technical Information Service
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TECHNICAL REPORT DATA *(Please read Instructions on the reverse before completing)*		
1. REPORT NO. EPA/600/J-84/365	2.	3. REC PB86-169893
4. TITLE AND SUBTITLE SCALING THE PHYSIOLOGICAL EFFECTS OF EXPOSURE TO RADIOFREQUENCY ELECTROMAGNETIC RADIATION: CONSEQUENCES OF BODY SIZE (Journal Version)		5. REPORT DATE 1984 6. PERFORMING ORGANIZATION CODE
7. AUTHOR(S) Christopher J. Gordon[1] and J. Homer Ferguson[2]		8. PERFORMING ORGANIZATION REPORT NO.
9. PERFORMING ORGANIZATION NAME AND ADDRESS [1]Phy. Sec., Bio. Eng. Brch., EBD, U.S. Environmental Protection Agency, Research Triangle Park, NC 27711 [2]Department of Biological Sciences, University of Idaho, Moscow, Idaho 83843, USA		10. PROGRAM ELEMENT NO. 11. CONTRACT/GRANT NO.
12. SPONSORING AGENCY NAME AND ADDRESS Health Effects Research Laboratory Office of Research and Development U.S. Environmental Protection Agency Research Triangle Park, NC 27711		13. TYPE OF REPORT AND PERIOD COVERED Journal Article 14. SPONSORING AGENCY CODE EPA - 600/11
15. SUPPLEMENTARY NOTES Int. J. Radiat. Biol., 1984, Vol. 46, No. 4, 387-397 October		

16. ABSTRACT

The authors

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17. KEY WORDS AND DOCUMENT ANALYSIS		
a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
18. DISTRIBUTION STATEMENT RELEASE TO PUBLIC	19. SECURITY CLASS *(This Report)* UNCLASSIFIED 20. SECURITY CLASS *(This page)* UNCLASSIFIED	21. NO. OF PAGES 12 22. PRICE

EPA Form 2220–1 (Rev. 4–77) PREVIOUS EDITION IS OBSOLETE

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EPA/600/J-84/365
JOURNAL ARTICLE

INT. J. RADIAT. BIOL., 1984, VOL. 46, NO. 4, 387–397

Scaling the physiological effects of exposure to radiofrequency electromagnetic radiation: consequences of body size

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(*Received 20 December 1983; revision received 15 May 1984; accepted 16 May 1984*)

We have demonstrated that a comparative analysis of the physiological effects of exposure of laboratory mammals to radiofrequency electromagnetic radiation (RFR) may be useful in predicting exposure thresholds for humans if the effect is assumed to be due only to heating of tissue. The threshold specific absorption rate (SAR) necessary to affect a thermoregulatory parameter shows an inverse and linear relationship to body mass. The inverse relationship between threshold SAR and body mass is attributed to a surface area : body mass relationship. In comparison to small mammals, relatively large mammals have a reduced capacity to dissipate an internal heat load passively, and are therefore physiologically more sensitive to RFR exposure. The threshold for a thermoregulatory response depends on the type of response measured, species, ambient temperature, etc. By extrapolation, it can be shown that a SAR of only 0·2–0·4 W/kg is required to promote a thermoregulatory response in a mammal with a body mass of 70 kg (e.g. weight of adult human). The specific absorption rate bioeffects data collected from laboratory mammals can be related by means of a simple power formula: threshold SAR (W/kg) = aM^b, where M is body mass in kg, a is a constant and b is equal to approximately $-0{\cdot}5$. Through this equation we have illustrated that a threshold SAR measured in a species weighing 100 g would be 10 times greater than that of a species weighing 10 000 g. Accordingly, a relatively low SAR that is physiologically ineffective in small mammals may be stressful to larger species.

Indexing terms: thermoregulation, microwaves.

1. Introduction

In 1966, an attempt was made to determine guidelines for limiting the exposure of humans to radiofrequency electromagnetic radiation (RFR) over the frequency range 10 MHz to 100 GHz (e.g. Mumford 1969). An incident power level of 10 mW/cm^2 was suggested as a standard of exposure. However, it was later recognized that at resonant RFR frequencies (e.g. for humans in free space 70–80 MHz), an incident power density of 10 mW/cm^2 would result in a specific absorption rate (SAR) of approximately 4·0 W/kg (Durney *et al.* 1978). Recent studies on the bioeffects of RFR exposure have shown that a SAR of 4·0 W/kg is usually high enough to cause adverse health effects (Cahill 1983, Tell and Harlen 1979). As a result, the newly proposed guideline of the American National Standards

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Institute (1982) suggests a human exposure limit of 300 kHz to 100 GHz at a SAR of 0·4 W/kg, which corresponds to an incident power level as low as 1·0 mW/cm^2 at frequencies of 70–80 MHz.

The literature on the bioeffects of RFR exposure indicates that a SAR of 0·4 W/kg is generally below the range of absorbed energy necessary to induce a physiological, pathological or behavioural response in relatively small laboratory mammals, such as mice, rats and guinea pigs (Cahill 1983). In these species, SARs of 1·0 W/kg and greater are generally necessary to induce a biological effect. For example, a SAR of 23 W/kg inhibits metabolism in mice (Ho and Edwards 1977), 5 W/kg increases blood levels of corticosterone in rats (Lu *et al.* 1981), and 7·0 W/kg offsets behavioural thermoregulation in mice (Gordon 1983 a). Exclusive exceptions to these observations are studies of the 'athermal' bioeffects of RFR exposure. For example, Blackman *et al.* (1979) found that frequency-modulated RFR exposure with a SAR equivalent to 0·0013 W/kg promotes changes in calcium efflux from chick brains. These data are difficult to interpret because no working hypothesis is adequate to explain the mechanism of the athermal effect, nor does an apparent dose–response relationship exist. Therefore, we have addressed only the data on the physiological response of mammals to 'thermalizing' levels of RFR in this paper.

Although partial body exposure data are available on the difference in thresholds for detecting infrared and radiofrequency radiation in humans (Vendrick and Vos 1958), few data on the whole-body effects of RFR exposure in humans exist. Ely and Goldman (1957) collected preliminary data on the effects of 2880 MHz in humans exposed at 240 mW/cm^2. At this level there was sweating and a slight drop in rectal temperature. Except for epidemiological studies, detailed information on the dose response of humans to whole-body resonant RFR exposure may never be collected. Therefore, in order to understand how humans might respond to RFR exposure, we must rely upon data collected on laboratory mammals.

Extrapolation of microwave effects from laboratory mammals to humans has been attempted (Michaelson and Schwan 1973, de Lorge 1979). A degree of uncertainty exists in predicting the response of humans to RFR exposure by extrapolating from the known bioeffects data collected on laboratory mammals. An obvious disadvantage to such extrapolation is the large difference in body size between experimental mammals and adult humans. For example, the mass of a laboratory rodent or a primate is 1·0–3·4 orders of magnitude less than the mass of a human (assuming an average adult male human body weight of 70 kg).

In some respects, the general physiological processes of laboratory mammals and humans differ markedly. A prime example is the physiological mechanism of heat dissipation during thermal stress. At high ambient temperatures, primates rely principally on sweating to dissipate excess body heat (Elizondo 1977), whereas rodents are unable to sweat but can dissipate heat through evaporative water loss from the skin and respiratory tract (Gordon 1982 a, b). Commonly used experimental primates, such as squirrel monkeys and rhesus monkeys, sweat when subjected to heat stress, but these mammals are considerably smaller than humans (e.g. 1 and 5 kg, respectively). Hence, physiological data collected on laboratory primates during RFR exposure may aid in predicting human responses, but the dose–response differences between humans and primates may be very large.

Computer models of the human response to RFR exposure are also useful for estimating the probable magnitude of the threshold SAR capable of activating physiological responses (Spiegel *et al.* 1980, Way *et al.* 1981). Such studies attempt

to compensate for the problems mentioned above by using appropriate physiological responses to heat stress in adult humans, for which abundant data exist, to model responses. The disadvantage in using these studies is that the physiological responses incorporated into the computer programs are those of humans exposed to high ambient temperatures and/or exercise, but not to RFR. Thus, these models can be used to calculate a dose–response relationship to RFR exposure, but only if the physiological responses are the same as those of humans subjected to natural forms of heat stress (e.g. exercise and/or radiant or convective heat). The similarity of response between RFR exposure and heat stress from exercise or exposure to high ambient temperatures is currently under debate.

In this paper we demonstrate that a comparative analysis of the bioeffects of RFR exposure on laboratory mammals may be useful in predicting SAR thresholds for exposed humans, if the effect is assumed to be due only to heating of tissue. Data on the physiological effects of RFR exposure have been collected on various laboratory mammals with body masses ranging from 0·02 to 7·0 kg, or a 350-fold increase in mass. If a relationship exists between an animal's mass and its sensitivity to thermalizing levels of RFR, then a regression analysis of the known physiological effects and their corresponding threshold SARs over such a wide span in body mass would allow a reliable extrapolation to a body mass of 70 kg. This would be an extrapolation of only 10-fold increase in mass and could give us an estimation of SAR thresholds for humans. A prediction of the absorbed RFR level necessary to activate a thermal physiological response in mammals of 70 kg will aid in our understanding of the responses of humans exposed to RFR.

2. Effect of body mass on thermal physiology of mammals

An animal's weight, mass and volume are proportional to the cube of its radial dimension, whereas its total surface area is proportional to the square of its radial dimension (Schmidt-Nielsen 1975). Thus, an animal's total surface area increases by approximately the 0·67 power with increasing body mass. Also, the surface area : mass ratio decreases logarithmically with increasing body mass. A regression line based on mammalian body mass versus surface area/mass has a slope of −0·33 (figure 1).

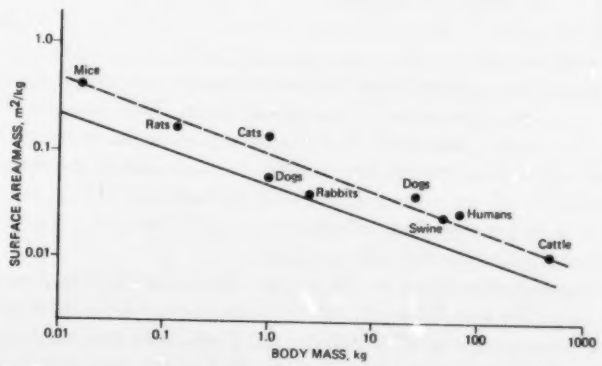

Figure 1. Relationship between the surface area: body mass ratio and the body mass of various mammals (Altman and Dittmer 1972). The continuous line represents the same relationship for a sphere of density 1·0. Linear regression equation: $\log y = \log 0{\cdot}087 - 0{\cdot}33 \log x$.

The importance of the surface area : mass ratio in the physiology of homeothermic animals can be illustrated by comparing the metabolism of two species with large differences in body mass, such as a 70 kg human and a 0·030 kg mouse. In figure 1, the surface area : mass ratio of a 30 g mouse is approximately 0·35 m^2/kg, while that of a 70 kg human is 0·025 m^2/kg. If both species maintain the same body temperature at a given ambient temperature, then the rate of heat loss from the body to the environment, normalized to body mass, is 14 times greater in mice than in humans. Therefore, metabolic activity in most mammals is related inversely to body weight.

The metabolic rate of mammals is a key example of a physiological function that decreases in activity with increasing body mass. Studies on mammalian metabolism have shown that the rate of heat loss as a function of body mass does not follow the surface area : mass relation intrinsically, but instead has a slightly less steep slope of −0·24 (Schmidt-Nielsen 1979; see figure 2, §4.1). This significant discrepancy between slopes has prompted numerous discussions (Schmidt-Nielsen 1972, Kleiber 1975). Regardless of the difference in slopes, the surface area : mass relationship has a direct effect on many physiological parameters in mammals regardless of species.

3. Equating heat of metabolism and absorbed heat from RFR exposure

For each millilitre of oxygen consumed in cellular metabolism, approximately 20·1 J or 4·8 calories of heat is liberated. Since all tissues consume oxygen, some to a greater extent than others, tissue throughout the body is continuously being heated internally at different rates. This thermal state is similar to that which occurs when an organism is exposed to thermalizing levels of RFR, especially at resonant frequencies that promote relatively deep heating. In a living organism, absorbed electromagnetic radiation is added to the organism's endogenous rate of metabolic heat production. Because whole-body oxygen consumption can be expressed in dimensions of power (W/kg), the time courses of metabolism and RFR absorption can be equated to the same dimensions.

A difficulty in equating the heating of RFR and metabolism is that RFR is absorbed unevenly throughout the body. Its accumulation in various regions of the body is based on morphology and chemical composition (Guy 1971), which would be quite distinct from the generation of metabolic heat. This is not to imply that metabolic heat is evenly generated. For example, the heart, kidneys, splanchnic organs and brain of a resting human have rates of heat production more than 10 times greater than those of other tissues and organs (Aschoff 1971). This would suggest that although heat is generated in different regions of the body, the same general principles of heat dissipation are still germane.

Below thermoneutral ambient temperatures, mammals can substitute RFR heat for metabolic heat (Ho and Edwards 1977). That is, metabolic rate will decrease when mammals are exposed to RFR radiation in a relatively cool environment. The following analysis is based on the assumptions that (a) the rate of heat production from metabolism and RFR exposure can be combined mathematically in the whole body in an additive or multiplicative fashion and (b) exposure occurs at a thermoneutral ambient temperature.

4. Physiological effects of RFR: consequences of body mass

4.1. *Theoretical evidence*

The metabolic rate versus body mass curve in figure 2 was calculated from the resting metabolic rate of mammals at a thermoneutral ambient temperature. For the following analysis, a 0·03 kg mouse and a 70 kg human are at a thermoneutral ambient temperature (~30°C), and each has a constant deep-body temperature of 37°C. (Although the body temperatures of these mammals may not be exactly 37°C, this approximation simplifies the following general calculations.)

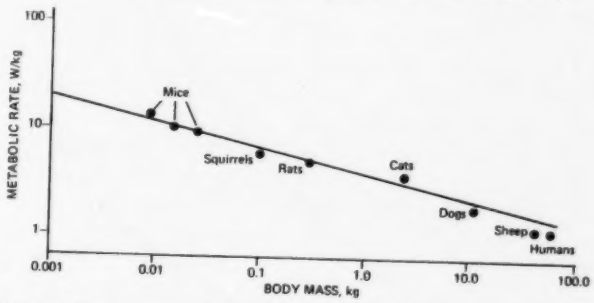

Figure 2. Relationship between the metabolic rates and body mass of mammals (Schmidt-Nielsen 1975). All measurements were made on resting animals under thermoneutral conditions. Linear regression equation; $\log y = \log 3{\cdot}88 - 0{\cdot}24 \log x$.

At thermoneutrality, the metabolic rate is 9·0 W/kg for the mouse and 1·39 W/kg for the human (figure 2). Thus, at the same ambient temperature, these two species use tremendously different rates of energy consumption to maintain their deep-body temperatures of approximately 37°C. This difference in energy expenditure is due primarily to the relative large surface area : body mass ratio of mice compared to that of humans.

If a mouse and a human are exposed to resonant RFR at a SAR of 1·0 W/kg, and both dissipate the excess heat load with appropriate thermoregulatory responses, the whole-body heat loss of the mouse will increase from 9·0 to 10·0 W/kg (an 11 per cent increase) while the human's heat loss will increase from 1·39 to 2·39 W/kg (72 per cent increase). Thus, based on the percentage change in whole-body heat loss, a small mammal will be less affected by a given SAR than a larger mammal.

The relationship between body mass and percentage increase in heat loss necessary to maintain normothermic temperature during exposure to RFR at SARs of 0·1, 0·4 and 1·0 W/kg is shown in figure 3. A SAR of 0·4 W/kg is included because this value represents the recently suggested exposure limit set by ANSI (1982). Because of the logarithmic relationship between surface area and body mass, the percentage heat loss increased logarithmically with body mass.

The relationship displayed in figure 3 provides a means of predicting the sensitivity (i.e. threshold SAR) of mammals exposed to RFR For example, assume that a thermoregulatory motor response is activated when the total heat gain (or heat loss assuming steady state conditions) from RFR esposure exceeds 25 per cent of the basal metabolic heat production. In this theoretical situation the threshold SAR would be calculated as:

$$\text{SAR} = [3{\cdot}88\, M^{-0{\cdot}24}](0{\cdot}25) \quad M = \text{mass in kg} \tag{1}$$

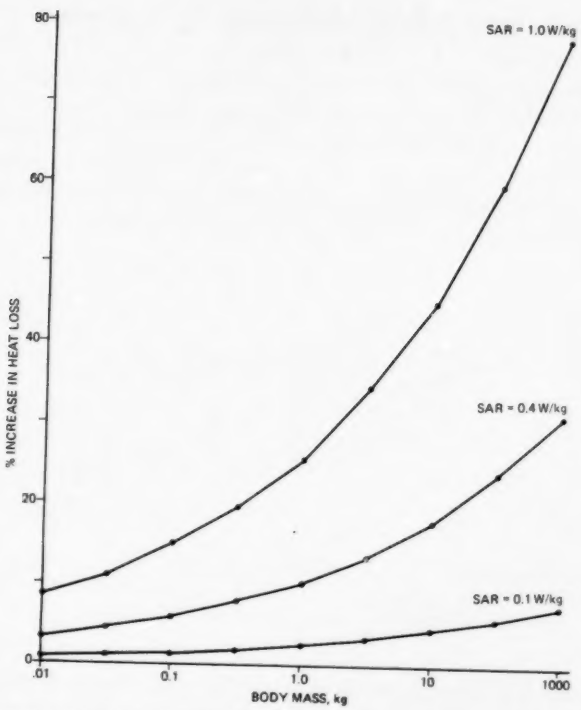

Figure 3. Relationship between mass and percentage increase in whole-body heat loss in mammals exposed to RFR at SARs of 0·1, 0·4 and 1·0 W/kg. The calculations are based on the assumption of resting metabolic rate at a thermoneutral ambient temperature, as in figure 2. Percentage heat loss calculated as (SAR/SAR + metabolic rate)*100.

The threshold SAR would be 3·0 W/kg for the 0·03 kg mouse and 0·46 W/kg for the 70 kg human. Note in the above equation that the term in brackets is the equation for metabolic rate as a function of body weight (figure 2). The 25 per cent total heat loss is used here only to demonstrate an idealistic situation. However, extensive work in squirrel monkeys exposed to 2450 MHz has shown that the threshold SAR for activating a thermoregulator response is approximately 15–25 per cent of the resting metabolic rate (Adair 1981). It is important to note that the threshold SAR that invokes a physiological response depends on ambient temperature, species, RFR frequency and type of response.

Using an approach similar to the foregoing analysis, Tell and Harlen (1979) derived a calculation, based on the basal metabolic rate (BMR), which predicted the SAR from RFR exposure that would raise the core temperature by 1°C. They concluded that at a frequency of 2450 MHz, humans and other animals would undergo a 1°C rise in core temperature when the thermal load from RFR exposure exceeded 1·5–2·0 times the BMR. Indeed, this implies an inverse relation between body mass and sensitivity to RFR For example, a 0·03 kg mouse exposed to 1·5–2·0 times its BMR would require a SAR of 13–18 W/kg to raise core temperature by 1°C whereas for a 70 kg human the SAR range would be 1·9–2·5 W/kg. Of course,

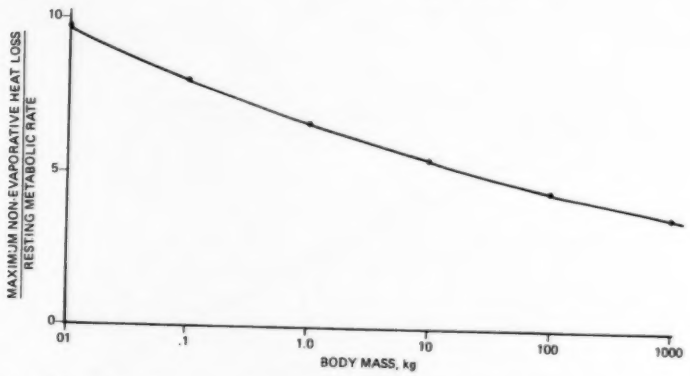

Figure 4. Effect of body mass on the maximum rate of non-evaporative heat loss relative to resting metabolism in mammals during exercise at an ambient temperature of 20°C (Taylor 1977). The calculations are based on the assumption that thermal conductance is a constant 1 cal $(cm^2\ hour\ °C)^{-1}$.

these values are dependent on critical environmental factors such as ambient temperature and relative humidity.

Data from exercise physiology research provide a valuable insight into the influences of body mass and ambient temperature on the threshold percentage heat loss necessary for activating a physiological response. In a comparative analysis of these data, Taylor (1977) calculated the maximum rate of passive heat loss or non-evaporative heat loss mammals can dissipate as a function of body mass. An inverse relationship was shown to exist between body mass and magnitude of non-evaporative heat loss (figure 4). For example, a 0·02 kg animal can dissipate 9 times its metabolic heat production with non-evaporative heat loss, whereas a 100 kg animal can dissipate only 4·5 times its metabolic heat production (assuming a 20°C gradient between body and ambient temperature). Of course, the thermal load from exercise and RFR exposure cannot always be equated physically or physiologically. If an increase in evaporative heat loss is used as a biological end-point, the total heat loss of a 0·02 kg animals would have to exceed 90 W/kg before it was observed, whereas that of a 100 kg species would have to exceed only 6 W/kg. Taylor's analysis shows that because of surface area : body mass relationships, small mammals can passively dissipate far greater amounts of heat than larger animals.

Ambient temperature is critical in determining a mammal's sensitivity to a heat load. For example, at an ambient temperature of 20°C, humans exercising at a work load of 40 W experience a 2·5 W/kg increase in heat production (and loss) and a slight elevation in evaporative heat loss. However, when the same work is performed at 30°C, the increase in evaporative heat loss is 5 times the magnitude of heat loss at 20°C (Chappuis *et al.* 1976).

4.2. *Experimental evidence*

It is not surprising, given the above analysis, that relatively high SARs are required to induce a physiological response in many laboratory mammals. Recently, Gordon (1982a) has shown that the approximate threshold SAR necessary to activate a physiological response, such as an increase in evaporative water loss, inhibition of metabolic heat production, increase in blood corticosterone or increase in heart and

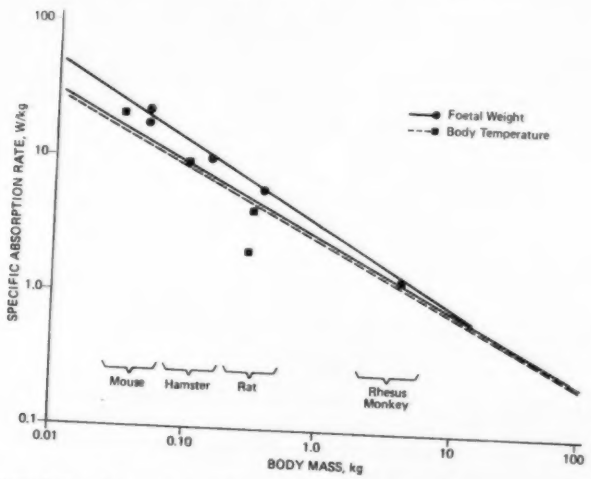

Figure 5. Correlation between threshold SAR and body mass for reduction in foetal weight of mice, hamsters and rats maternally exposed to 2450 MHz, and a 0·5–1·0°C increase in body temperature in mice, hamsters and rats exposed to 2450 MHz and rhesus monkeys exposed to 225 MHz. Ambient temperatures of all studies ranged from 20 to 24°C. Body temperature data taken from Lu *et al.* (1981), Lotz and Michaelson (1978) and Lotz (1982). Foetal weight data taken from Berman *et al.* (1978, 1981, 1982), and Berman and Carter (1983). The regression line for predicting threshold SAR developed by Gordon (1982 a) is plotted for comparison (lighter continuous line, see eqn. (2)). Linear regression equation for foetal mass, or $\log y = \log 3{\cdot}46 - 0{\cdot}53 \log x$; for body temperature, $\log y = \log 2{\cdot}45 - 0{\cdot}52 \log x$.

respiratory rate, is inversely proportional to body mass (figure 5). The relationship between the threshold SAR necessary to affect a physiological parameter and the body mass was found to be linear on a double logarithmic scale, and described by the equation:

$$\text{threshold SAR (W/kg)} = 2{\cdot}75\, M^{-0{\cdot}53} \quad M = \text{mass in kg} \tag{2}$$

The selection of the data by Gordon (1982 a) was based on the criterion that the physiological parameters were directly or indirectly involved in an animal's normal response to heat stress. Despite the wide variation in ambient temperatures (20–35°C), RFR frequencies, species and physiological parameters measured, the strong statistical significance of the regression line ($r = 0{\cdot}9407$), $P < 0{\cdot}005$) implied that body mass plays a critical role in determining the threshold SAR It is significant that the regression line of best fit is linear on a double logarithmic scale, which was also true for surface area : weight versus body mass (figure 1) and metabolic rate versus body mass (figure 2). Because of this logarithmic relationship, extrapolation of a threshold SAR to scale the experimental data to the body mass of a larger mammal is probably also logarithmic. For example, based on equation (2), a threshold SAR of 20 W/kg in a 0·03 kg species is predicted to be equivalent to 4·0 W/kg in a 0·3 kg species, and 0·25 W/kg in a 70 kg species.

It is recognized that the data used to construct the regression line by Gordon (1982 a) were collected by different investigators under different ambient conditions. However, the exactness of fit of these data into a single regression line ($r = -0{\cdot}94$)

lends powerful evidence that the phenomenon of surface/volume relationship attenuates the effect of other factors on threshold SAR of a species.

Several investigators have measured the relationship between the SAR and a rise in body temperature in mammals maintained at a relative narrow ambient temperature range of 20–24°C. A double logarithmic plot of the SAR required for a 0·5–1·0°C rise in core temperature versus body mass (figure 5) yields a linear relationship that is very similar to the data of Gordon (1982 a). Extrapolation of the data in figure 5 indicates that the SAR necessary to raise body temperature in a 70 kg mammal by 0·5–1·0°C is predicted to be 0·25 W/kg.

The data for rhesus monkeys in figure 5 (Lotz 1982) are important because exposure occurred at a near-resonant frequency of 225 MHz. Resonant exposure, in which the energy is deposited very deep in the body, may be a worst-case situation. The mouse data in figure 5 were also collected at resonant frequencies (~2450 MHz). For a 0·03 kg mouse exposed to resonant RFR at an ambient temperature 22°C, a SAR of 22 W/kg is required to raise the body temperature 0·5–1°C. For a comparable temperature rise in a 4 kg rhesus monkey at an ambient temperature of 24°C, a SAR of only 1·2 W/kg is required. Thus, assuming that large species follow a similar pattern to small species, an animal having 10 times the mass of the rhesus monkey exposed to a resonant RFR would be expected to undergo a similar rise in temperature at a SAR less than 1·0 W/kg.

Berman *et al.* (1978, 1981, 1982) and Berman and Carter (1983) determined the minimal SAR necessary to promote a 10 per cent reduction in the body mass of foetuses in pregnant mice, hamsters and rats exposed for 100 min/day to 2450 MHz microwave radiation at an ambient temperature of 20–22°C. Reduction in mass of the foetus is thought to occur when microwave exposure increases tissue temperature to foetotoxic levels. The lowest observed SAR for a reduction in foetal mass versus the body mass of the exposed mammal is shown in a double logarithmic plot in figure 5. Like the body temperature data, the data collected on the pregnant mammals are close to the threshold SAR predicted by the general regression line of Gordon (1982 a).

The data from figure 5 indicate that with increasing mass the efficacy of absorbed R.F. radiation to raise body temperature is enhanced. Generally speaking, all thermoregulatory effectors (e.g. behaviour, peripheral vasomotor tone, evaporation) are activated by increasing body temperature. Hence, the threshold SAR for activating a physiological effector is most likely to decrease with increasing SAR Recently, Gordon (1983 b) has demonstrated an inverse correlation between body mass and the threshold SAR for RFR-induced inhibition of metabolic rate and peripheral vasodilation for mammals ranging in mass from 0·03 to 1·0 kg. The threshold SARs for raising ventilatory frequency (Gordon and Long 1984) and activating behavioural selection of a cooler ambient temperature in the mouse and hamster (Gordon *et al.* 1984) are also related inversely to body mass. It remains to be seen if these relations can be extrapolated to larger species, especially humans (Adair *et al.* 1983).

4.3. *Threshold verses tolerance*

The primary objective of this paper is to demonstrate the relationship between body mass and the threshold level of SAR that promotes a thermoregulatory response. Tolerance of mammals to RFR exposure is another matter entirely. However, many investigators have attempted to calculate the highest tolerable SARs

or those that evoke strong thermoregulatory responses (Durney *et al.* 1973, Tell and Harlen 1979, Schwan 1982). Both the tolerable limits of exposure and the minimum level at which the subject is affected are important.

5. Conclusion

Body mass and surface area have been considered as principal independent variables that might allow one to relate the RFR bioeffects between experimental animals and humans (Michaelson and Schwan 1973, Durney *et al.* 1978). In the present study we have attempted to demonstrate the importance of the surface area mass ratio on the physiological sensitivity of mammals to RFR.

The linear responses in figure 5 indicate that SARs of approximately 0·2–0·4 W/kg would be sufficient to promote a 0·5–1·0°C rise in body temperature in a mammal with a body mass of 70 kg. This predicted threshold value is no doubt affected by numerous environmental conditions, such as ambient temperature, relative humidity, species (e.g. comparison of furred versus bare-skinned mammals) and, especially, RFR frequency. Resonant frequencies that deposit energy deep inside the body might be expected to stress physiological systems to a greater extent than relatively high frequencies absorbed predominantly in the peripheral tissues (D'Andrea *et al.* 1977, Lotz 1982). Thus, dissipation of a heat load by passive conductance is easier if the heat is absorbed on the periphery. For example, when exposed to a near-resonant frequency of 225 MHz, rhesus monkeys undergo a 0·5–0·6°C increase in rectal temperature at a SAR of 1·2 W/kg. However, at a supraresonant frequency of 1290 MHz, a SAR of 3·0 W/kg is required to induce the same temperature elevation (Lotz 1982).

Because of potential health hazards, restrictions have been imposed on the study of human thermoregulatory response to RFR exposure. Thus, past physiological studies have been limited to subhuman primates, rodents and other laboratory mammals. This paper demonstrates that the SAR dose–effects data collected on laboratory mammals may be related to the probable threshold responses of larger animals such as humans. Because of the inverse, logarithmic relationship between body mass and threshold SAR, experiments conducted on humans would be expected to yield lower threshold SARs than experiments conducted on species smaller than humans. On the other hand, a relatively low SAR that has no physiological effect in small rodents may be highly stressful when applied to a member of a larger species such as an adult human.

Acknowledgments

This paper has been reviewed by the Health Effects Research Laboratory, U.S. Environmental Protection Agency, and approved for publication. Mention of trade names of commercial products does not constitute endorsement or recommendation for use. Opinions expressed in this paper are those of the authors and do not necessarily reflect official EPA opinion. We thank Drs E. Berman and R. Smialowicz of EPA and M. Galvin and D. McRee of NIEHS for their review of an earlier draft of the manuscript.

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